AMENDMENTS TO THE SPECIFICATION:

Please replace the Sequence Listing filed on January 13, 2004 with the Substitute Sequence Listing submitted herewith.

Please replace paragraph [00307] with the following amended paragraph:

Soluble FcγRIIIA and FcγRIIIB fusion proteins were generated by fusing the extracellular regions of each receptor to the constant region of IgG2. The extracellular domain of human FcγRIIIA was fused to the hinge-CH2-CH3 (*i.e.*, hinge-constant region) region of human IgG2 (this construct will be referred to as "sFcγRIIIA-G2"; SEQ. ID. NO. 6). The IgG2 constant region was chosen because the FcÿR does not bind to this constant region. The extracellular region of FcγRIIIA was amplified using a full length cDNA clone as template (gift from Dr. Jeffrey Ravetch, Rockefeller University) using the following primers: SJ45f (CTC TCC ACA GGT GTC CAC TCC ATG CGG ACT GAA GAT CTC CCC; SEQ ID NO: 10); and SJ48r (GCG CTC GAC TTG GTA CCC AGG TGG; SEQ ID NO: 11). This amplified fragment was then joined to a signal sequence coding segment (source of signal sequence: Synthetic construct made at MacroGenics based on mouse genomic segment coding for a VH signal sequence and intron, GENBANK Accession No. M12880; Primers: H009 (CGA GCT AGC TGA GAT CAC AGT TCT CTC TAC; SEQ ID NO: 12); SJ27r (GGA GTG GAC ACC TGT GGA GAG; SEQ ID NO: 13))and to the IgG hinge-CH2-CH3 (amino acids 216-446) segment by an overlapping PCR procedure. Source of the IgG2 constant region was PMGX101: cDNA clone made at MacroGenics, and the primers were SJ47f (CCT GGG TAC CAA GTC GAG CGC AAA TGT TGT GTC GAG TGC CC; SEQ ID NO: 14) and SJ20r (GGC GAA TTC GCG GCC GCA CTC ATT TAC CCG GAG ACA GG; SEQ ID NO: 15). The resulting fragment was digested with NheI and EcoRI and cloned into the mammalian expression vector pCI-neo. The inclusion of the hinge region allows flexibility of the two receptor arms and covalent disulfide linkage of the each monomer.

Please replace paragraph [00309] with the following amended paragraph:

Primers for amplification were SJ84f (GGC GGC TAG CCA CCA TGG GAA TCC TGT CAT TCT TAC C; SEQ ID NO: 16); and SJ82r (CAT TTG CGC TCC CCC ATG GGT GAA GAG CTG GGA GC; SEQ ID NO: 17) and joined to the hinge-CH2-CH3 cDNA of human IgG2 constant region by overlapping PCR. The resulting fragment was digested with NheI and EcoRI and cloned into the mammalian expression vector pCI-neo.

Please replace paragraph [00331] with the following amended paragraph:

Several mutants in this region were constructed, 156GSKNV160 (SEQ ID NO: 43) --> GYTLF (SEQ ID NO: 44), V160F, and 154LV155 --> NI.

Please replace paragraph [00336] with the following amended paragraph:

114 -->DKP) which lacked 3G8 binding but retained Fcγ binding capacity. They were pursued further for animal studies. Mutant 156GSKNV160 --> GYTLF which lacked both immune complex binding and 3G8 binding was also expressed for using in assay development as a negative control.

Please replace Table 3 on page 112 with the following amended Table 3:

TABLE 3. BINDING CHARACTERISTICS AND IMMUNE COMPLEX BINDING OF MUTANTS

		IC Binding	FcγR inhibited by 3G8	3G8 Binding
WT	Mutants	+++	+++	+++
pMGX327	156GSKNV160 --> GYTLF	-	-	-
pMGX328	V160F	+++	+	-
pMGX329	154LV155-->NI	+++++	++	+/-
pMGX330	112NTA114-->DKP	+++++	+	+/-
pMGX331	H116V	-	-	+++
pMGX335	V155F	+	+	+
pMGX336	Y137H	+++++	+++	+++
pMGX337	G126D	-	-	-
pMGX338	N112D	+++	+++	+++
pMGX339	T113K	+++++	-	+++++
pMGX340	A114P	+++++	++++	++++

Please replace paragraph [00338] with the following amended paragraph:

The construct sFcγRIIIA-G2 was mutagenized in order to generate soluble FcγRIIIA fusion proteins with enhanced stability. The wild type sFcγRIIIA-G2 had limited stability as assessed by SDS-PAGE analysis. In order to improve the stability of the FcγRIIIA-G2 fusion protein, four variants were generated (FIG. 1A). Analytical results suggested that the source of the instability was proteolysis at or near the junction of the FcγR and the Fc segments, which corresponds to amino acids S183-S184. In humans, proteolysis near the C-terminus of the extracellular domain, at residues V196-S197, (Galon *et al.* 1998, *Eur. J. Immunol.* 28: 2101-7; numbering is in accordance to SEQ ID. NO. 1) has been demonstrated to result in the release of the naturally occuring receptor from cells into a soluble form, *see, e.g.*, Galon *et al.* 1998, *Eur. J. Immunol.* 28: 2101-7; Fleit *et al.*, 1992, *Blood*, 79: 2721-8; Masuda *et al.* , 2003, *J. Rhematol.*

30(9): 1911-7; Masuda *et al.*, 2003, *Clin. Exp. Immunol.* 132(3): 477-84, all of which are incorporated herein by reference in their entireties. Cell bound FcγRIIB, however, is not subject to such naturally occurring proteolysis *in vivo*. In two variants of FcγRIIIA-G2, V1 and V2, the C-terminus of the extracellular domain, LAVSTISSFFPPGYQV (SEQ ID NO: 45), was replaced by a flexible GGGGS (SEQ ID NO: 46) linker sequence. In two other variants, V3 and V4, the C-terminus of FcγRIIIA residues ITQGLAVSTISSFFPPGYQV (SEQ ID NO: 47) was replaced by the equivalent segment of FcγRIIB, VQAPSSSPME (SEQ ID NO: 48). An additional difference between FcγRIIIA and FcγRIIB in this region is the presence of an N-linked glycosylation site in FcγRIIIA which is not present in FcγRIIB. Thus, in V1 and V3 the FcγRIIIA sequence at this position was retained while in V2 and V4 the non-glycosylated FcγRIIB sequence was utilized. These subsequences are depicted in FIG. 1A (FcγRIIIA-G2 wild type, SEQ ID NO. 49; V1, SEQ ID NO: 50; V2, SEQ ID NO: 51; V3, SEQ ID NO: 52; V4, SEQ ID NO: 53; and FcγRIIB-G2, SEQ ID NO: 54. These molecules were constructed by an overlapping PCR method and expressed transiently in HEK-293 cells.